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# Effect on lipid profile in mice experimentally infected and vaccinated with Aspicularis tetraptera

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**Abstract**

**Aspicularis tetraptera worm (nematode) has been shown to affect the lipid profile (triglyceride, cholesterol, HDL, and LDL) of mice infected and vaccinated with somatic antigen. This study was designed to investigate this effect. After vaccination with somatic antigen, lipid profile values returned to normal in mice with a severe infection. The most effective antigens have been found to be those derived from egg somatic cells, according to research.**

**Keywords**

**Triglyceride, Cholesterol, HDL, LDL, Lipid Profile, Aspicularis tetraptera.77**

## Introduction

There are numerous medical and economic applications for helminths, which are multicellular parasitic organisms. Infectious parasites of helminth have been detected in humans and domesticated animals all over the world. Intriguingly, parasites (helminths) have been shown to live as adults for up to two years [1]. One of the most significant causes of illness and death, particularly in underdeveloped nations [2], was helminths. More than 2.5 billion people have parasitic worm infections, according to a research by the World Health Organization [3]. Children are more likely to be infected with intestinal helminths [4]. An estimated 57 percent of the human population (developing countries) may be afflicted by helminths by the year 2025. Infectious disorders transmitted by helminths are a major cause of decreased productivity in animals. Most of this can be attributed to lower mortality and reduced weight growth, among other things. In tropical regions, where rainfall is plentiful, this problem is particularly acute [9-10]. The Aspicularis oxyurid nematode, a common parasite of rodents, is seen frequently. The fact that rodent pinworms persist despite control measures points to a problem with the diagnostics and eradication processes. Many institutions are unable to offer effective treatment for pinworm eradication because of problems in identifying parasite presence [11]. Laboratory rodent parasites include the oxyurids (Pinworm), Syphcia muris, Syphcia obvelata, and Aspicularis tetraptera. Pinworms have been shown to impact weight and growth, as well as the intestines on one side [12, 13, 14, 15, 16, 17] [12, 13, 14, 15, 16, 17, 18]. A concern in the treatment of helminthic disorders is

the development of resistance among intestinal helminths to anthelminthic medications [18]. Given the significance of the problem, the current study examined the effect of somatic antigen on the lipid profile level in infected and vaccinated mice. It was shown that

## Materials and Method

### Experimental animals

The present study utilized Mus musculusalbinus, a strain of albino Swiss mice. Pharmacists at IPS Academy in Indore loaned these animals, which were housed under normal lighting, airflow, and temperature conditions. For this study, only healthy male mice aged 7-8 weeks old and weighing 30-50 gms were used. Mice's feces confirmed the findings. Every day, the mice were given water and a balanced meal in sterilized cages.

### Maintenance of Aspicularis tetraptera strain:

the Parasitology Laboratory of Zoology at Govt. Holkar Science College in Indore, where Aspicularis tetraptera was originally obtained (M.P). After every 31st day of infection, Aspicularis tetraptera is routinely maintained in the laboratory by a dose of 100 varied embryonated eggs. The parasite's numerous life stages were obtained from the affected animals and used in the research. It was detailed by Wakelin [19] how Aspicularis tetraptera is maintained, infected, and recovered.

### Preparation and infection of eggs-inoculums:

Before administering any dose, the first step was to count the embryonated eggs. Next, the dose was prepared by dissolving 100 eggs in 2.0 ml of solution. Finally, the dose was administered orally via an attached feeding needle.

Finally, experimental mice were placed in different cages as per the experimental design after they had been inoculated with the virus.

Cages of mice were used and standard diet was given to them. e)

### Preparation of somatic antigens:

a) Homogenization and lyophilization are among the methods used.

All phases of development, from eggs to adults, were properly cleaned with distilled water before storage.

b) The eggs, larvae, and adults were all homogenized separately in protein-free culture media.

Finally, each homogenate was lyophilized and stored at a temperature of 4 degrees Celsius.

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**Blood collection and serum separation:**

This was accomplished by dissecting the heart of an experimental mouse.

b) A 2ml glass syringe was used to draw blood from the ventricle.

c) Blood was withdrawn from the syringe and placed in a 15-ml centrifuge tube.

Because of this, these tubes had to be maintained in the freezer.

e) There was evidence of clotting in the test tube.

f) Serum was pipetted out of these tubes and kept at 20°C. Estimation of lipid profile

**Estimation of plasma total cholesterol**

Allain's enzymatic approach was used to measure total cholesterol.

**Estimation of Plasma High Density Lipoprotein (HDL)**

To assess HDL cholesterol, a spectrophotometer reading at 545 nm was used with a redox test kit [20] to compare the absorbances of the sample controlled and the standard.

Estimation of Plasma Triglycerides

Triglycerides in plasma were evaluated using glycerol peroxidase-oxidase technique [21]. The intensity of the coloured chemical produced is measured at 545nm using spectrophotometer.

**Estimation of Plasma Low Density Lipoprotein (LDL)**

Using the phosphor-tungstate magnesium chloride technique, we were able to obtain the LDL-cholesterol concentration.

**Result**

Mice infected with Aspicularis tetraptera and vaccinated with various concentrations of egg somatic antigen had their lipid profiles measured on the 31st day after infection, and the results are summarized in the tables and figures (1, 2, and 3). (1, 2 and 3).

**Total Lipid (in mg/dl)**

NINVC1 and INVC2 had total lipid values of 579 mg/dl and 438 mg/dl, respectively, in the current investigation. IVEgSoAg1, IVEgSoAg2, IVEgSoAg3, IVEgSoAg4, and IVEgSoAg5 infected and vaccinated animals had total lipid levels of 465 mg/dl, 510 mg/dl, 540 mg/dl, 545 mg/dl, and 552 mg/dl, respectively, of A. tetraptera somatic egg antigen in the serum.

450 mg/dl in IVLSoAg1 and 495 mg/dl in IVLSoAg2, 510 mg/dl in IVCSoAg3, 525 mg/dl in IVLSoAg4, and 532 mg/dl in IVLSoAg5 were the total lipid values observed in A. tetraptera infected and vaccinated mice, respectively, with varied concentrations of somatic antigen of Larvae.

Total lipid levels in A. tetraptera infected and vaccinated mice were 440 mg/dl in IVASoAg1, 482 mg/dl in IVASoAg2, 498

mg/dl in IVASoAg3, 505 mg/dl in IVASoAg4, and 515 mg/dl in IVASoAg5 with varied concentrations of adult worm somatic antigen.

In comparison to NINVC1, the total lipid levels in INVC2 were found to be lower. However, when the concentration of somatic antigen was raised, total lipid values were shown to rise. Based on somatic antigen levels, we were able to determine the observed values (Tables 1, 2, and 3& figures 1, 2, and 3).

**Cholesterol (In mg/dl)**

123 milligrams per deciliter were found in NINVC1 and 94 milligrams per deciliter were found in INVC2. A. tetraptera-infected and vaccinated mice had cholesterol levels of 101 mg/dl in IVEgSoAg1, 109 mg/dl in IVEvSoAg2, 116 mg/dl in IVEvSoAg3, 119 mg/dl in IVEvSoAg4, and 122 mg/dl in IVEvSoAg5.

All of the IVLSoAg concentrations showed 98 mg/dl of cholesterol in the A. tetraptera infected and vaccinated with IVLSoAg1, 108 mg/dl in IVLSoAg2, 115 mg/dl in IVLSoAg3, and 120 mg/dl in IVLSoAg5.

When Aspiacularis tetraptera were exposed to different levels of adult somatic antigen, the cholesterol levels were found to range from as low as 90 mg/dl in IVASoAg1 to as high as 111 mg/dl in IVASoAg5 in the presence of IVASoAg1 and as high as 97 mg/dl in IVASoAg2 in the absence of IVASoAg1.

Compared to NINVC1, cholesterol levels in INVC2 were found to be lower. When the concentration of somatic antigen increased, researchers saw a rise in cholesterol levels. Somatic antigen concentration was closely linked to cholesterol levels.

Cholesterol levels ranged from 122 mg/dl in the IVASoAg1 group, which had the highest levels, to 90 mg/dl in IVEgSoAg5. Maximum cholesterol value of egg somatic antigen.

**Low density lipoprotein (LDLin mg/dl)**

A total cholesterol level of 73.3 mg/dl was found in NINVC1 while a level of 51.45 mg/dl was found in INVC2.

Infected and vaccinated A. tetraptera mice with varying concentrations of egg somatic antigen had LDL levels of 58.4 mg/dl in IVEgSoAg1, 60.2 mg/dl in IVEgSoAg2, 65.5 mg/dl in IVEgSoAg3, 66.4 mg/dl in IVEgSoAg4, and 68.3 mg/dl in IVEgSoAg5 and 68.3

A. tetraptera-infected and vaccinated mice with varied concentrations of larvae somatic antigen showed low-density lipoprotein values of 54.3 mg/dl, 57.2 mg/dl, 60.2 mg/dl, 64.4 mg/dl, and 65.6 mg/dl.

Mice infected with A. tetraptera and vaccinated with varying concentrations of adult worm somatic antigen showed low

density lipoprotein values of 45.2 mg/dl in IVASoAg1.

52 mg/dl in IVASoAg3, 56.4 mg in IVASoAg4 and 60.2 mg/dl for the fifth and final sample, respectively.

LDL levels were found to be lower in INVC2 than in NINVC1. Somatic antigen concentration was observed to enhance LDL levels. As the concentration of somatic antigens increased, the values increased as well (tables1, 2, and 3and figures 1, 2, and 3).

The highest LDL levels were found in the IVEgSoAg5 group, at 68.3 mg/dl, while the lowest levels were found in the IVASoAg1 group, at 45.2 mg/dl. In egg somatic antigen, the highest LDL values were found.

**High Density Lipoprotein (HDL in mg/dl)**

NINVC1 had a high-density lipoprotein value of 36 mg/dl, while INVC2 had a value of 28.5 mg/dl.

With varying concentrations of egg somatic antigen, HDL levels in A. tetraptera infected and vaccinated mice ranged from 30 mg/dl in IVEgSoAg1, 30 mg/dl in IVEgSoAg2 to 34 mg/dl for the A. tetraptera-vaccinated mice.

HDL levels were found to be 29 mg/dl in IVLSoAg1, 31 mg/dl in IVLSoAg2, 31 mg/dl in IVLSoAg3, 33 mg/dl in IVLSoAg4, and 34 mg/dl in IVLSoAg5 in mice infected with A. tetraptera and vaccinated with varied concentrations of larvae somatic antigen.

A. tetraptera-infected and vaccinated mice with varied concentrations of adult worm somatic antigen showed HDL values of 26, 28, 29, and 30 mg/dl, respectively.

In INVC2, HDL levels were observed to be lower than in NINVC1. However, when the concentration of somatic antigen was raised, HDL values rose. The amount of somatic antigen correlated directly with the amount of HDL that was produced (tables 1, 2, and 3 and figures 1, 2, and 3).

IVEgSoAg5 and IVLSoAg5 had the highest HDL values of 34 mg/dl and 26 mg/dl, respectively, whereas IVASoAg1 had the lowest.

**Table-1**

**Lipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen of eggs.**

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| Group No. | Groups | Total lipid mg/dl | Cholesterol mg/dl | Low density lipoprotein (LDL) mg/dl | High density lipoprotein (HDL) mg/dl |
|---|---|---|---|---|---|
| 1. | $NINVC_1$ | 579 ±3.834 | 128 ±2.280 | 73.3 ±0.698 | 36 ±2.828 |
| 2 | $INVC_2$ | 438 ±4.295 | 94 ±2.828 | 51.45 ±0.664 | 28.5 ±1.612 |
| 3 | $IVEgSoAg_1$ | 465 ±3.405 | 101 ±2.828 | 58.4 ±0.282 | 30 ±0.7071 |
| 4 | $IVEgSoAg_2$ | 510 ±0.894 | 109 ±2.607 | 60.2 ±0.070 | 30 ±0.368 |
| 5 | $IVEgSoAg_3$ | 540 ±1.414 | 116 ±3.847 | 65.5 ±3.479 | 32 ±1.581 |
| 6 | $IVEgSoAg_4$ | 545 ±4.939 | 119 ±3.84 | 66.4 ±0.2 | 32 ±0.894 |
| 7 | $IVEgSoAg_5$ | 552 ±1.414 | 122 ±1.414 | 68.3 ±0.2 | 34 ±1.414 |

| | |
|---|---|
| **$NINVC_1$** | Non infected non vaccinated control-1 |
| **$INVC_2$** | Infected non vaccinated control-2 |
| **$IVEgSoAg_1$** | Infected vaccinated with 20µg eggs somatic antigen. |
| **$IVEgSoAg_2$** | Infected vaccinated with 40µg eggs somatic antigen. |
| **$IVEgSoAg_3$** | Infected vaccinated with 50µg eggs somatic antigen. |
| **$IVEgSoAg_4$** | Infected vaccinated with 80µg eggs somatic antigen. |
| **$IVEgSoAg_5$** | Infected vaccinated with 100µg eggs somatic antigen. |

**Figure-1**

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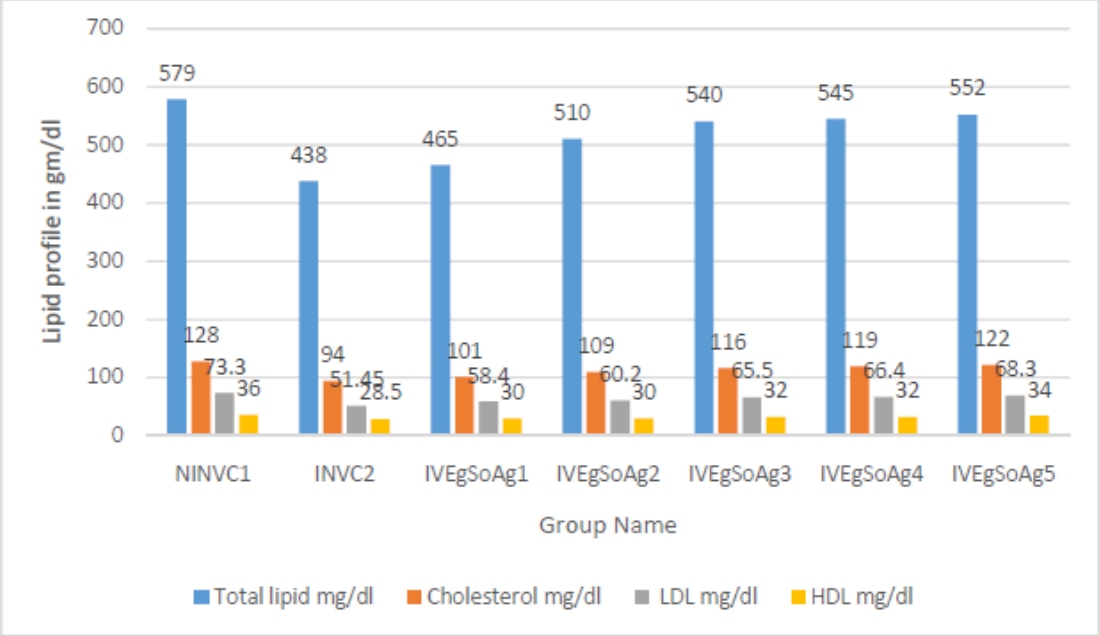
**ShowingLipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen of eggs.**




| | |
|---|---|
| **NINVC$_1$** | Non infected non vaccinated control-1 |
| **INVC$_2$** | Infected non vaccinated control -2 |
| **IVEgSoAg$_1$** | Infected vaccinated with 20µg eggs somatic antigen. |
| **IVEgSoAg$_2$** | Infected vaccinated with 40µg eggs somatic antigen. |
| **IVEgSoAg$_3$** | Infected vaccinated with 60µg eggs somatic antigen. |
| **IVEgSoAg$_4$** | Infected vaccinated with 80µg eggs somatic antigen. |
| **IVEgSoAg$_5$** | Infected vaccinated with 100µg eggs somatic antigen. |

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**Table-2**

**Lipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen of larvae.**

| Group No. | Groups | Total lipid mg/dl | Cholesterol mg/dl | Low density lipoprotein (LDL) mg/dl | High density lipoprotein (HDL) mg/dl |
|---|---|---|---|---|---|
| 1. | $NINVC_1$ | 579 ±3.405 | 128 ±1.414 | 73.3 ±0.698 | 36 ±2.366 |
| 2 | $INVC_2$ | 438 ±4.73 | 94 ±2.828 | 51.45 ±0.664 | 28.5 ±1.673 |
| 3 | $IVLSoAg_1$ | 450 ±3.405 | 98 ±1.414 | 54.3 ±0.384 | 29 ±0.824 |
| 4 | $IVLSoAg_2$ | 495 ±3.687 | 108 ±4.472 | 58.2 ±0.469 | 31 ±3.847 |
| 5 | $IVLSoAg_3$ | 510 ±1.266 | 115 ±2.607 | 60.2 ±3.289 | 31 ±1.414 |
| 6 | $IVLSoAg_4$ | 525 ±0.282 | 116 ±3.687 | 64.4 ±0.316 | 33 ±1.41 |
| 7 | $IVLSoAg_5$ | 532 ±0.635 | 120 ±2 | 65.5 ±0.352 | 34 ±3.687 |

| | |
|---|---|
| **$NINVC_1$** | Non infected non vaccinated control-1 |
| **$INVC_2$** | Infected non vaccinated control-2 |
| **$IVLSoAg_1$** | Infected vaccinated with 20µg larvae somatic antigen. |
| **$IVLSoAg_2$** | Infected vaccinated with 40µg larvae somatic antigen. |
| **$IVLSoAg_3$** | Infected vaccinated with 50µg larvae somatic antigen. |
| **$IVLSoAg_4$** | Infected vaccinated with 80µg larvae somatic antigen. |
| **$IVLSoAg_5$** | Infected vaccinated with 100µg larvae somatic antigen. |

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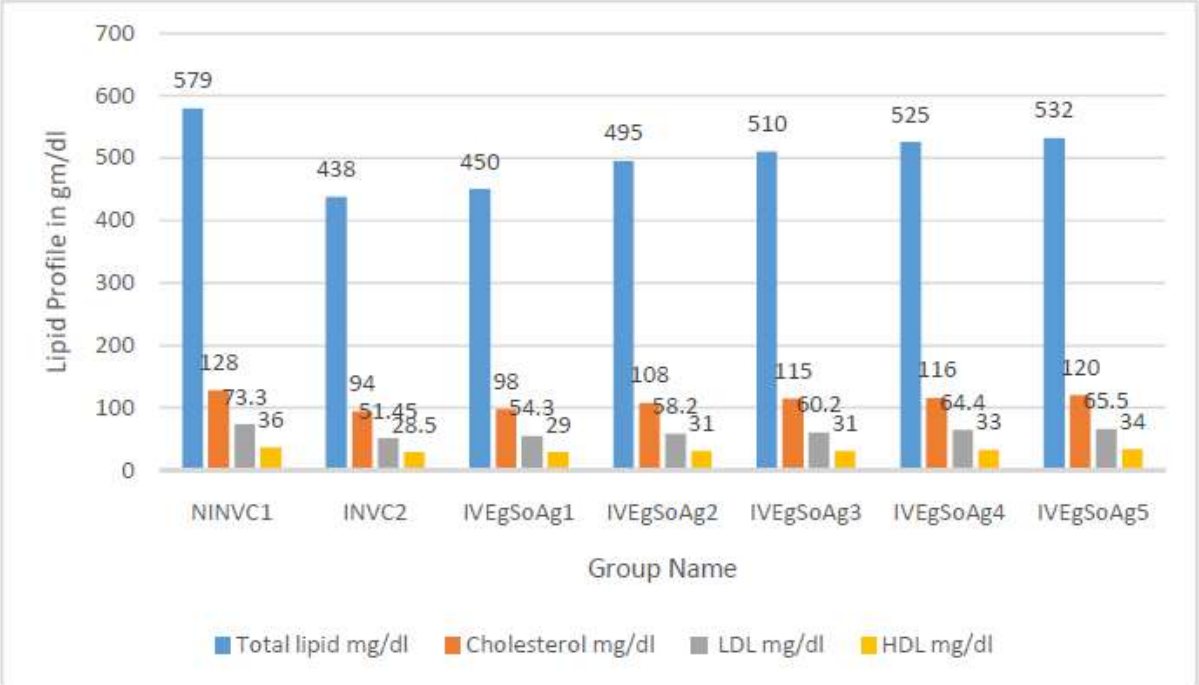
**Figure-2**

**ShowingLipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen of larvae.**




| | |
|---|---|
| **$NINVC_1$** | Non infected non vaccinated control-1 |
| **$INVC_2$** | Infected non vaccinated control -2 |
| **$IVEgSoAg_1$** | Infected vaccinated with 20µg eggs somatic antigen. |

| | |
|---|---|
| **IVEgSoAg$_2$** | Infected vaccinated with 40µg eggs somatic antigen. |
| **IVEgSoAg$_3$** | Infected vaccinated with 60µg eggs somatic antigen. |
| **IVEgSoAg$_4$** | Infected vaccinated with 80µg eggs somatic antigen. |
| **IVEgSoAg$_5$** | Infected vaccinated with 100µg eggs somatic antigen. |

**Table-3**

**Lipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen of antigen of adult worm.**

| Group No. | Groups | Total lipid mg/dl | Cholesterol mg/dl | Low density lipoprotein (LDL) mg/dl | High density lipoprotein (HDL) mg/dl |
|---|---|---|---|---|---|
| 1. | NINVC$_1$ | 579 ±4.195 | 128 ±1.414 | 73.3 ±0.698 | 36 ±1.788 |
| 2 | INVC$_2$ | 438 ±4.836 | 94 ±2.828 | 51.45 ±0.664 | 28.5 ±1.124 |
| 3 | IVLSoAg$_1$ | 440 ±4.0496 | 90 ±3.224 | 45.2 ±0.322 | 26 ±3.224 |
| 4 | IVLSoAg$_2$ | 482 ±1.266 | 97 ±0.920 | 48.4 ±0.316 | 28 ±0.322 |
| 5 | IVLSoAg$_3$ | 498 ±0.644 | 103 ±2.828 | 52.8 ±0.572 | 29 ±0.141 |
| 6 | IVLSoAg$_4$ | 505 ±2.607 | 107 ±2.607 | 56.4 ±2.151 | 30 ±1.279 |
| 7 | IVLSoAg$_5$ | 515 | 111 | 60.2 | 30 |

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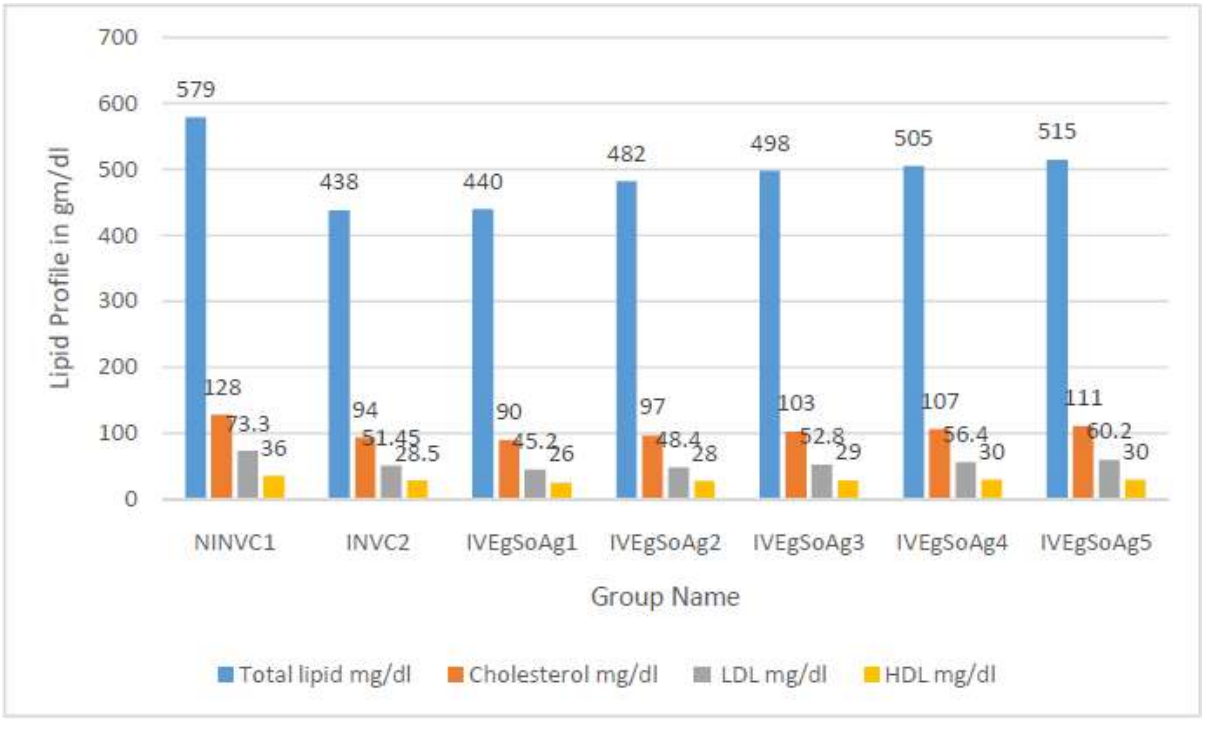
| | | | | | |
|---|---|---|---|---|---|
| | | ±0.6 | ±2 | ±0.447 | ±0.850 |

| | |
|---|---|
| **$NINVC_1$** | Non infected non vaccinated control-1 |
| **$INVC_2$** | Infected non vaccinated control-2 |
| **$IVLSoAg_1$** | Infected vaccinated with 20µg larvae somatic antigen. |
| **$IVLSoAg_2$** | Infected vaccinated with 40µg larvae somatic antigen. |
| **$IVLSoAg_3$** | Infected vaccinated with 50µg larvae somatic antigen. |
| **$IVLSoAg_4$** | Infected vaccinated with 80µg larvae somatic antigen. |
| **$IVLSoAg_5$** | Infected vaccinated with 100µg larvae somatic antigen. |

**Figure-3**

**ShowingLipid Profile values taken on 31st day post infection from mice infected with A. tetraptera and vaccinated with different concentrations of somatic antigen adult worm.**



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| | |
|---|---|
| **$NINVC_1$** | Non infected non vaccinated control-1 |
| **$INVC_2$** | Infected non vaccinated control -2 |
| **$IVEgSoAg_1$** | Infected vaccinated with 20µg eggs somatic antigen. |
| **$IVEgSoAg_2$** | Infected vaccinated with 40µg eggs somatic antigen. |
| **$IVEgSoAg_3$** | Infected vaccinated with 60µg eggs somatic antigen. |
| **$IVEgSoAg_4$** | Infected vaccinated with 80µg eggs somatic antigen. |
| **$IVEgSoAg_5$** | Infected vaccinated with 100µg eggs somatic antigen. |

**Discussion**

Tables (1, 2 and 3) describe the lipid profile results in infected and vaccinated mice with varying concentrations of somatic antigen, and the figures show the results (1, 2 and 3). A. tetraptera-infected mice had lower total lipids, cholesterol, LDL and HDL compared to control mice, who were not parasitized. It's possible that the lower lipid profile value was caused by parasites in the digestive tract eating lipid content. Somatic antigens may have killed parasites and expelled them from the host, increasing total lipid cholesterol in infected and vaccinated mice (i.e. experimental animals treated with varying concentrations of somatic antigen). According to this study, the host's gastrointestinal tract was free of parasites after vaccination with a somatic antigen (somatic antigen). The lipid profile was shown to have decreased as a result of infection. This could be the result of hepatic dysfunction, in my opinion. As a result, an improvement in the lipid profile in the vaccinated mice may be an indicator of healing processes. The presence of parasites has also been linked to the release of hormones and liver dysfunction [22, 23]. B. malayi-infected livers showed a decrease in lipid content, according to Joshi [24]. In addition, Wiedermann [25] found lower serum lipid levels in the Hookworm, Strongyloides, and Trichuris-infected Shipibo community (Peru). Camels infected with nematodes were also tested for total cholesterol, triglyceride levels, HDL, LDL, and VLDL levels (Strongyloides, Trichuris, Tichostrogylidae). Lipid levels, on the other hand, have dropped significantly. According to these scientists, a decrease in lipid levels could be the result of parasites feeding on the lipids in the host's bloodstream.

**Conclusion**

In this study, mice infected with Aspicularis tetraptera had lower lipid profile values (total lipid, cholesterol, LDL, and HDL) than animals immunized with somatic antigens of egg,

larvae, and adult worm (at varied concentrations).

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Bibliography

1. The principal of Govt. Holkar Science College, Dr. Suresh T. Silawat, deserves special recognition for providing the necessary laboratory equipment.

2. This study was conducted by Colley DG; Savioli LP; and LoVerde PT by Colley DG (2001). Infectious disease The field of helminthology in modern medicine. Science,293:1437-1438.

3. 3. A. Mulla Wahid, V.S. Thorat, R.V. Patil, and K.B. Burade (2010). AlocasiaIndica leaf anthelmitic activity Pharm Tech Research International. Pharmaceutical Technology Research, 2:26-30

The Immunology of Parasites 36 (2014): 439-452 (Zaph C, Cooper PJ, Harris NL 2014).

4. C.A.N. Clewes and C. Shaw (2000). British Medical Bulletin, vol. 56, no. 1, pp. 193-208, January 2000.

5. 5. Sorobetea, D., Svenssonfreg, M., and Grencis, R. Sorobetea (2018). Gastrointestinal nematode infection is prevented. Volume II: 304-315 of Mucosal Immunology.

6. Affirmative action is a key component of the Sustainable Development Goals (SDG) (1994). The impact of helminths on tropical African small ruminant output. Medical Journal of the Republic of Kenya.

7. This study was conducted by seven researchers: Gatongi; Scott; Rajan; Gathum; Munyua; Cheruiyot; and Prichard (1997). Antihelminthic treatment regimes for sheep and goats in semi-arid Kenya: effects on flock performance. 68(4): 323-336, Veterinary Parasitol.

8. 8. Radostitis, O.M., Leslie K. and Fetrow E. Radostitis (1994). J: Sheep health and management of productivity. Food Animal Production Medicine, Second Edition, Herd Health

9. The W.B. Saunders Company

10. 9. WHO helminthiasis report (2012). Number of children treated for soil-transmitted helminthiasis in 2010. No. 23, 87:225-232, Weekly Epidemiological Record.

11. T.M. Meade and J. Watson (2014). The study of red pinworm (Syphaciamuris) epidemiology as a means of increasing detection and elimination of the parasite. Scientific Reports, 53(6): 661-667.

12. 12. Hoag, W.G. (ed) (1961). Oxyuriasis in mouse colonies in the laboratory. In the American Journal of Veterinary Research, 22:150-153.

13. 14. Harwell, J.F. and Boyd, D.D.'s research (1968). In mice, oxyuriasis is a normal occurrence. The Journal of the American Veterinary Medical Association 153: 950-953.

14. Flynn, R.J. (1973). Parasites of laboratory animals, Ames: Iowa State University Press.

15. Pearson, D.J. and Taylor, G. (1975). The influence of the nematode Syphaciaobvelata

on adjuvant arthritis in rats. Immunology. 29:391-396.

16. Taffs, L.F. (1976). Pinworm infections in laboratory rodents: a review. Lab Anim Sci.10:1-13.

17. Wagner, M. (1988). The effect of infection with the pinworm (Syphaciamuris) on rat growth. Lab. Anim. Sci. 38:476-478.

18. Nery, P.S., Nogueir, F.A., Martins, E.R., Duarte, E.R. (2010). Vet Parasitol.,171, 361-364.

19. Wakelin, D. (1967). Acquired immunity to Trichurismurisin the albino laboratory mouse. Parasitol. 57: 515-524.

20. Lopes, M.F., Virella. (1997). ClinChem, 23, 882.

21. Buccolo, G., David, I. (1973). ClinChem, 19, 476-482.

22. Silveria, A.M., Friche, Aa. and Rumjanek, F.D. (1986). Transfer of (14C) cholesterol and its metabolites between adult male and female worms of Schistoma mansoni. Comp. Biochem Physiol B. 85: 851-57.

23. Biadun, W. (1990). Studies of serum lipids in guinea pig with larval ascariasis. Wiad Parazytol, 36: 15–26.

24. Joshi, A., Saxena, J.K., Murthy, P.K., Sen, A.B. and Ghatak, S. (1989). Brugia malayi

status of host during different stages of infection. Folia Parasitol, 36: 169 - 75.

25. Wiedermann, U., Stemberger, H., Unfried, E., Widhalm, K., Kundli, M., Altenriederer, M., Savedr, M. and Wiedermann, G. (1991). Intetinal worm burden and serum cholesterol or lipid concentration in Shipibo population (peru). Zentrabl Bakteriol, 275: 29-86.

26. Mohamed, A.M., Mohmoud, R., Ellah, A. and Ghada Abou El-Ella A. (2008). The Influence of some nematode parasitism on lipid metabolism and lipoprotein profile in Dromedary Camel (Camelus dromedarius). Journal of Camelid Sciences, 1: 63-67.